surgery. The chest operations involved either an extensive thoracotomy with open drainage (two patients) or a thoracotomy with excision of the diseased or abnormal tissue (eleven patients).

2. Vital capacity determinations and measurements were made of the chest wall in the antero-posterior and transverse diameters, and the circumferences of all patients before and at the end of each treatment.

3. Each patient was treated 30 minutes daily for one week or more. Restriction of the excursion of the normal side of the chest was accomplished by means of sand bags and towels placed between the chest wall and the anterior shell of the plastic cage. Patients were instructed to force breathe while synchronizing the rate and rhythm with the alternate phases of compression and rarefaction of the air within the plastic shell of the respirator.

4. Twelve of the thirteen showed an increase in the antero-posterior diameter of the chest at the level of the manubrium; nine at the level of the nipple line; six at the level of the ninth rib.

5. All patients showed a disappearance of the lag of the anterior chest wall on the operated side, the excursion of the chest wall on the two sides becoming equal and synchronous.

6. All patients stated that they felt better at the end of the treatment period, and there was a disappearance of any signs of dyspnea which may have been present at the beginning of the treatment.

7. It is concluded that the Blanchard Mechanotherapist provides a beneficial type of treatement which may be used to assist in establishing normal pulmonary ventilation and in reexpanding the lung in patients having residual postoperative atelectasis following surgery.

Fatal Myocarditis With Complete Heart Block From Diphtheria

R. A. KOCHER, M.D., *Carmel*

DIPHTHERIA, as is well known, if not treated promptly and sufficiently with antitoxin may cause serious myocardial damage. Such cases are now rarely encountered and this is remarkable when we note that less than two generations ago Aschoff[1] reported that 10 to 20 per cent of all cases of diphtheria showed parenchymatous degeneration of the heart muscle. This improved condition may be credited to the early recognition of the disease and the prompt treatment with antitoxin. Of 60 patients with diphtheria treated with antitoxin at the 203rd General Hospital, United States Army, during 1944-45, none showed any clinical evidence of heart involvement.

Outside this series, a German prisoner of war with diphtheria, for which no antitoxin had been given (he had been taken prisoner on the sixth day of the disease), entered our service at a stage when grave damage to the heart already had occurred. Because of the severity of the heart damage, manifest by clinical symptoms and the series of electrocardiographic tracings characteristic of heart block, later confirmed by post-mortem examination, this case is here reported.

CASE REPORT

A German prisoner of war, 29 years old, was admitted to the hospital with complaints of pain in the left cervical region, swollen neck glands and inability to swallow. Six days before admission he first developed sore throat for which he used pills and gargles. Twenty-four hours after onset, swelling in the neck began and became progressively worse until it became very painful to swallow. He was in seriously ill condition when picked up by American troops and sent to the hospital.

On admission there was intense edema of the soft palate and left posterior pillar. The posterior pharyngeal space was greatly inflamed and frank pus was present. Because of the appearance of the throat the admitting officer entered the patient on the surgical service where an incision was at once made between tonsil and capsule on the left. Diphtheria was not suspected and no smear or culture was taken on the day of admission.

On the day following admission the patient showed bradycardia, a pulse rate of 42 and temperature of 36.3 C. The throat now appeared covered with a gray gangrenous exudate over left tonsil and pillar. A throat culture was taken at this time and the patient was transferred to the medical service. The blood pressure was 85 systolic, 70 diastolic; respiration slow and often sighing. On this same

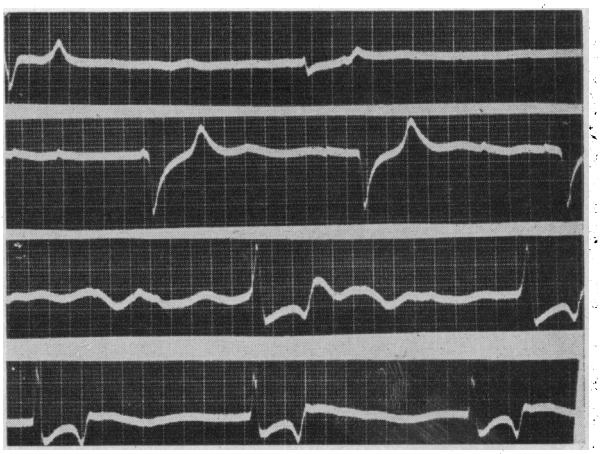

Fig. 1.—Electrocardiogram, four leads; showing complete dissociation of auricular and ventricular complexes. Auricular rate 110, regular; Ventricular rate 30, irregular. Eighth day from onset of diphtheria.

day the electrocardiograph (Fig. 1) showed complete heart block; ventricular rate 30, auricular rate 110. On the third day after admission, or ninth day after onset of illness, the pulse became weak and rapid—130 at the wrist. The electrocardiograph (Fig. 2) now showed ventricular tach-

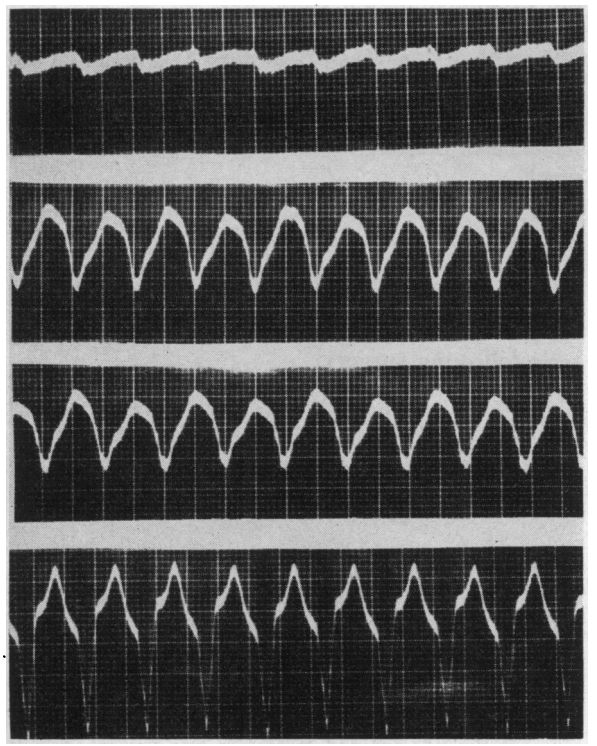

Fig. 2.—Electrocardiogram, four leads; showing paroxysmal ventricular tachycardia. Ventricular rate 150, regular. Ninth day from onset of dyphtheria.

ycardia; rate 150, regular. Fluid intake recorded for this day was 1,150 cc. and output 50 cc. Temperature was 36.2 C. The throat culture at this time was positive for C. diphtheriae. Administration of quinidine sulphate intravenously, 0.2 gm. every three hours, was begun at 5 P.M. The following morning the tachycardia was still present and another electrocardiographic tracing (Fig. 3) was made at 10 A.M. The rate was still 150 with regular rhythm. Quinidine was continued until a total of 1.2 gm. had been given. By 4:30 in the afternoon the pulse had dropped to 78 with regular rhythm and sounds of fair

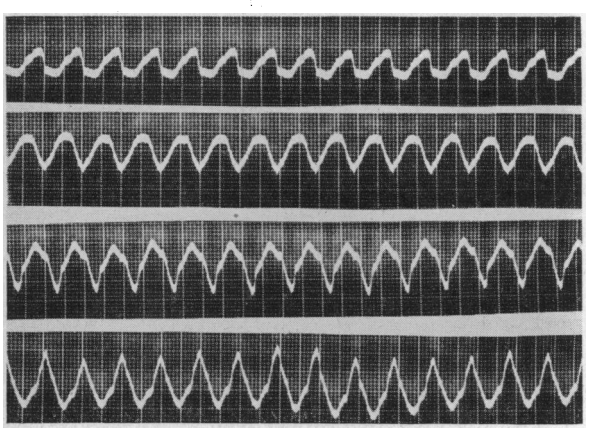

Fig. 3.—Electrocardiogram, 4 leads; on second day of ventricular tachycardia; tenth day from onset of disease. 10 A.M.

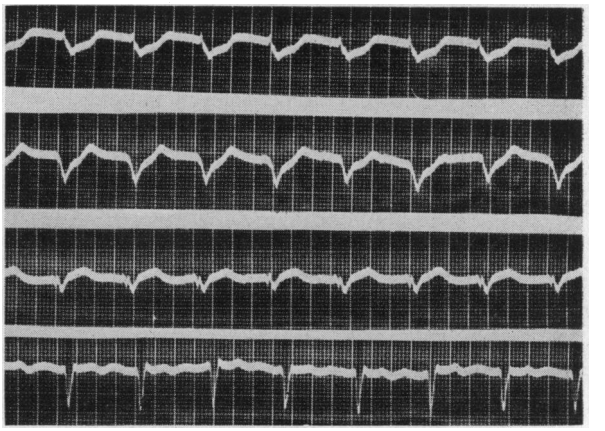

Fig. 4.—Electrocardiogram, 4 leads; twenty hours after beginning use of quinidine sulphate. (Total 18 gr.). Ventricular rate 85, regular. 5 P.M. 11th day from onset of disease. Of special interest is lead four in which the ventricular complexes are regular and fairly normal and the possibility that here we have a transient auriculo-ventricular nodal rhythm. Though no P waves are apparent, this could be accounted for by the action of quinidine in depressing the reversal of the impulse through the junctional tissue into the auricles. The ventricular rate of 85 is somewhat against this supposition.

quality at the apex. An electrocardiograph (Fig. 4) was taken at 5 P.M. The patient's condition became progressively worse and he died during the night at 1:15 A.M.

Laboratory reports: On the day of admission, R.B.C. was 4,300,000; Hg. 90 per cent; W.B.C. 9,100; Segs. 61, stabs 2, eosin. 3, lymphs. 33, monos. 1. Urinalysis showed Sp. Gr. 1,028; no albumin, no sugar.

Clinical diagnosis: diphtheria, pharyngeal; myocarditis with heart block; paroxysmal ventricular tachycardia.

AUTOPSY FINDINGS

An autopsy was performed by Major George J. McHeffey. The pericardial sac contained 70 cc. clear straw colored fluid. The heart weighed 465 grams and was somewhat increased in transverse diameter. The epicardial covering was smooth and glistening except for a few scattered petechial hemorrhages. On opening the heart the right ventricle was found to be markedly dilated. There was also a moderate dilatation of the left ventricle. The valves were thin, smooth and translucent. The tricuspid valve measured 14 cm., the pulmonic valve 7.5 cm., the aortic valve 6.4 cm., the mitral valve 8.5 cm. The thickness of the right ventricle measured 0.5 cm. and that of the left ventricle 2 cm.

On sectioning the heart, numerous small areas of fibrosis were seen, but a more striking feature was the presence of alternate areas of red and yellow discolorations suggesting acute infarction. However, the coronary arteries were patent and there was a minimal amount of arteriosclerosis.

Microscopic examination of the heart septum revealed a diffuse destruction of heart muscle fibers (Fig. 5.), the destroyed fibers taking on a deep blue stain suggestive of acute necrosis. Surrounding the splintered and destroyed fibers were diffuse and local infiltrations of neutrophylles, hymphocytes, monocytes and a few plasma cells. There was also a moderate leukocytic exudate invading the muscle fibers that were destroyed. This destructive process extended into the junctional tissue between

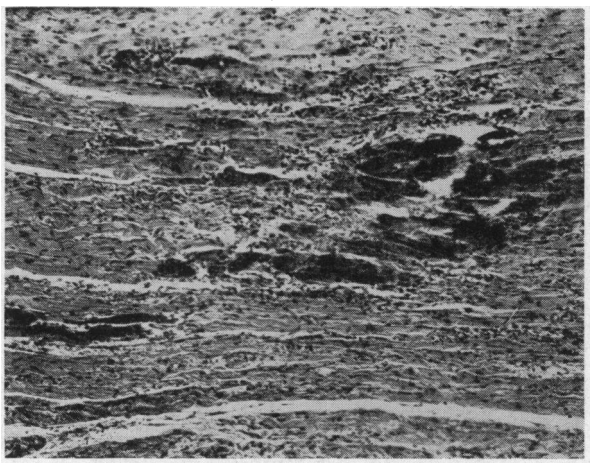

Fig. 5.—Photomicrograph showing acute diphtheritic myocarditis. Extensive destruction and necrosis of muscle fibers is apparent. Also diffuse and local infiltrations of neutrophyles, lymphocytes, and formative cells.

auricles and ventricles as well. In other areas the inflammatory exudate was perivascular and associated with perivascular fibrosis.

The pathological diagnosis was (1) Diphtheria, laryngeal, tracheal. (2) Myocardial degeneration, toxic. (3) Hydrothorax, bilateral. (4) Pleuritis, right. (5) Bronchopneumonia, bilateral. (6) Hydropericardium.

Clinically, the striking feature in this case is the high grade of break in a-v conduction, manifest by severe bradycardia, later by paroxysmal tachycardia, and the electrocardiographic tracings characteristic of complete heart block. Further, the restorative, though futile, of normal rate following the use of quinidine sulphate. These striking clinical findings were found at postmortem examination to be due to equally striking pathological changes in the myocardium.

It should not pass without comment that had the diphtheria been recognized in the early stages instead of after the lapse of seven days from onset of symptoms, antitoxin in adequate dosage might have prevented this damage to the heart muscle, hence might have been life saving. It is unlikely that this would have availed on the day of admission, the sixth of the disease, when the patient first fell into American hands, although no excuse is offered for not taking smears and cultures at that time.

Diphtheria, as well as certain other infectious diseases—rheumatic fever for example—in those occasional cases where they produce milder grades of heart block from the action of a soluble toxin, commonly produce a transient effect from which complete or nearly complete recovery is the rule. In these cases that recover, the condition causing interference with conduction is probably transient perivascular cellular infiltration with edema. "In diphtheria the conductive system is invaded by lymphocytes and eosinophiles. These collections tend to be perivascular and may be so extensive as to disrupt the continuity of the bundle. The muscle is edematous and where not replaced by cellular elements is swollen, granular and stains poorly."[3] Sir Thomas Lewis[2] in discussing heart block from infection states: "In most such infections, however, the block is transient, there being recovery from much or all of the original damage." Paul White and T. Ducket Jones[4] made a follow-up study of 100 consecutive individuals who had proven severe diphtheria from five to eight years prior to examination. Quoting from this study, Paul White[5] reports: "There is little chronic effect on the heart from diphtheria, even when it is severe. Survival usually means escape from any permanent or severe heart disease. Slight lesions may perhaps persist which cannot be discovered by any careful study years afterward. . . . Not one of these 100 cases showed any evidence of heart disease." On the other hand, there is the occasional and now rare severe and fatal case such as that here reported, and White[5] also makes the statement, "Undoubtedly death during diphtheria results from myocardial involvement in a considerable percentage of fatal cases."

In the present instance it soon became apparent clinically that the break in protoplasmic continuity was complete so that conduction was no longer possible. The action of quinidine in restoring normal rate from a ventricular tachycardia of 150 is not altogether clear. Offhand one would hesitate to use this drug in the presence of heart block because of its depressing effect on a-v conduction. However, from the electrocardiogram (Fig. 3), we see a type of tachycardia evidently arising in the a-v node with p waves buried in the QRS complexes. One of the actions of quinidine is to lengthen the refractory period of heart muscle (from 50 to 100 per cent), and it would seem most likely that this accounts for the effect in slowing the rate in the present instance.

In our patient the findings on postmortem examination were parenchymatous degeneration of the heart muscle, involving not the a-v bundle alone, but almost all the heart muscle as well. And this diffuse involvement is also consistent with the general rule that complete a-v block is almost invariably evidence of more extensive damage to the heart muscle.

SUMMARY

A case of complete heart block from diphtheria in a 29-year-old male, resulting fatally, is presented. The salient clinical and pathological features are presented.

REFERENCES

1. Aschoff, L.: Pathologische Anatomie, 1909, Vol. 2, p. 130, Gustav Fischer, Jena.

2. Lewis, Sir Thomas: Diseases of the Heart, 3rd Ed., 1943, Macmillan Co. Ltd., London.

3. Nomenclature and Criteria For Diagnosis of Diseases of the Heart. 1945, New York Heart Association, New York.

4. White, Paul D., and Jones, T. D.: The Heart After Severe Diphtheria, American Heart Journal, 3: 190, 1927.

5. White, Paul D.: Heart Diseases, 3rd Ed., 1945, The Macmillan Co., New York.